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SERIES AND FAMILIES OF DISEASES

Recent medical literature swarms with titles in which two or more diseases, seemingly disparate, are linked together in a causal relation. The view that certain diseases may exist in series or families, oftentimes pivoting around some cutaneous affection, such as an erythemas or a purpura, is of comparatively recent origin, but was already implicit in the essential tenets of the Coan or Hippocratic school of medicine. Those who have thought most deeply on this theme are Hippocrates, Sydenham, the nosologists of the 18th century, Charcot, Allbutt, Osler, the dermatologists (Bazin, Stephen Mackenzie), and the recent English school of epidemiologists (Crookshank). The initial distinction between the Coan and Cnidian schools of medicine turns, be it said, upon the possibility of so separating the essentials of diseases from their accidents that we can recognize them unmistakably as in the Hippocratic Canon; or of labelling and classifying them in a minute and meticulous way, as in the extant fragments of Cnidian medicine. Hippocrates aimed at prognosis, with very generalized therapy; the Cnidians at diagnosis, with specific therapy. In the view of Littré, Hippocratic medicine was right enough in tendency for the 5th century, B. C., when instrumental diagnosis and refined therapy were unknown and non-existent. Cnidian medicine is the medicine of our own period of functional tests and highly specialized therapeutics. In the view of the eminent classical scholar Jones, Hippocrates was great in spite of his method. He separated medicine from priestcraft and philosophy, viewed and treated the patient as a whole, left unapproachable pictures of phthisis, malarial fever, epidemic parotitis, puerperal septicæmia and epilepsy (without labels), devised a method of wound treatment that was virtually

aseptic and gave us the lofty code of ethics implicit in the "Oath" and the "Law." The Cnidians, on the other hand, misapplied what Jones regards as a "truly scientific method."[1] They mistook symptoms for diseases, described three kinds of tetanus and four kinds of consumption. The culmination of the tendency is apparent in the 191 varieties of syphilis described by Brassavola in the 16th century. Until very recently, fever, tremor, chorea, and paralysis were regarded as diseases rather than symptoms, and to-day we are wrestling with the anæmias, the jaundices, the rheumatic, gouty, pseudo-typhoid and pseudo-diphtheritic affections, the mixed infections, the pluriglandular syndromes and the baffling modalities of such major diseases as syphilis, tubercle or influenza. "The Cnidians," in the view of Jones, "were comparatively unsuccessful because they had not learned to distinguish the essential from the unessential. Hippocrates (he says) was a genius who followed a will-o'-the-wisp; the Cnidians were plodders along the dreary stretch of road that lies before every advance in knowledge. Hippocrates did the wrong thing well; the Cnidians did the right thing badly. . . . The truth seems to be that the peculiarly Hippocratic doctrines are of greater interest and value to scientists than they are to practising doctors." But in cases where insulin or salvarsan fail, where the physician is baffled by "the dim and half-seen signals of disease," the truth is perhaps the other way around. It is a fact, however, that the tendency of medicine in our own time has been essentially Cnidian—the medicine of the bacteriologist, the serologist, and the pathologist, rather than that of the bedside doctor. The minutiæ of diagnosis and therapy have become more important than the patient himself.

In the 17th century, Sydenham, although a devotee of the Hippocratic or humoral pathology, inclined to the erroneous Cnidian view that each disease constitutes a definite species, classifiable as a botanist classifies plants. Although he made no special classification himself, Sydenham was, in theory at least, a forerunner of the 18th-century nosologists, who beginning with Linnæus (1763), arranged diseases after the botanical method. The most remarkable of these was Cullen, who divided diseases into fevers, neuroses, cachexias and local disorders, differentiated

[1] W. H. S. Jones: Hippocrates, London, 1923, II, pp. XIV–XIX.

thirty-four kinds of chronic rheumatism and regarded even gout as a neurosis. Ludicrous as this view of gout may seem at first sight, it acquires a peculiar interest in the light of Charcot's investigations of gout and rheumatism. In the Tuesday Lectures of Charcot, (December 6, 1887),[2] the relation between gout and the neuroses is set forth in a novel light:

"We may regard arthritism as a tree, of which the principal branches are gout, articular rheumatism, certain forms of migraine, cutaneous affections, etc. On the other hand, there is a neuropathic tree, comprising neurasthenia, epilepsy, all insanities, hereditary or other, general paralysis, locomotor ataxia, etc. The two trees exist, side by side, communicating by their roots, and with relations otherwise so intimate that they may sometimes be mistaken for one and the same tree. With this key, you will understand most of the phenomena which come to pass in nervous diseases; without it, you will be unable to explain them. In dealing with a nervous patient, you should regard the malady before you as merely an episode. Thus, in a case of chorea, it is only necessary to inquire how long it has existed. The condition of the patient is only an accident in the history of the disease, just as each of us is only an accident in the history of humanity."

The immediate starting point of this line of thought was the teaching of such men as John Brown and particularly Broussais, whose greatest service to medicine was to dispense with the metaphysical or ontological conception of disease as a "clinical entity." Indeed, twenty years before Charcot's pronouncement, Clifford Allbutt, fresh from the clinics of Trousseau and Bazin, made a bold assault upon the fictitious notion of "clinical entities" with reference to the classification of skin diseases (1867).[3] The motto of his article is a citation from Broussais, denouncing the vicious tendency to regard pathology as a science independent of the actual diseases it deals with.[4]

[2] Charcot: Lecons du Mardi, Paris, 1887–8, 39.

[3] T. C. Allbutt: St. George's Hosp. Repts., Lond., 1867, II, 187–204.

[4] The extent to which this false view of pathology has gained ground in the popular imagination is indicated in H. L. Mencken's view of the pathologist as a terrier sniffing at ratholes. It is right and proper, however, that anatomy, physiology and pathology should be given separate places in public libraries, apart from medicine, as these basic disciplines are also correlated with general biology and its branches, with primitive plastic imagery and with the fine arts in general.

Allbutt points out at the start, that to regard a disease as a circumscribed clinical entity is no better than the savage's view of a disease as a particular "indwelling devil," nor do such later variants as "morbid principle," "virus," "bacillus" tell the whole story. He penalizes particularly such notions as "types of disease" or "typical cases," as being applied ordinarily to express, not the assemblage of differential characters common to a whole group (its lowest common denominator), but the logical opposites, *e.g.*, a striking individual or "classical" specimen of some group, with which all other cases are to be squared, "regardless of the fact that nature never makes two leaves on the same tree alike." Thus in the case of epilepsy, there are many forms of motor spasm, apart from the classical fit with loss of consciousness, and many of these are associated with other affections, *e.g.*, syphilis, tubercle, gout and the psychoneuroses. The skin, in particular, is a most effective pivot for the classification of diseases in general, as being the essential *fundamentum divisionis* in Huxley's classification of races of men and as being associated with four of the main diatheses, *viz.*, the syphilitic, tubercular, arthritic and dartrous (herpetic). Gouty, tubercular, luetic and scrofulous patients have complexions all their own, as well as specific cutaneous eruptions. Advancing a step further, Allbutt points out that life itself is to the physician a series of physiological processes, of which scrofula, rheumatism, periods of good health, syphilis, tubercle, rickets, etc., may be regarded as phases or "modes of growth." Such morbid states as bronchitis, meningitis, lichen, eczema, cardiac or renal disease are usually members or terms of such series, although commonly diagnosed and treated in practice as independent, self-limited diseases. Viewed from the Coan or Hippocratic angle, eczema is not a disease but "a member of a series having terms preceding and succeeding." Viewed from the Cnidian angle, it is a genus comprising several species, "each of which has its place in its own series." Eczema may have its stages of pityriasis, psoriasis or lichen, but eczema arthriticum or eczema diabeticorum are truer expressions of classification than eczema erythematosum or lichenoid eczema, which are merely superficial transient phases. The merit of Willan's classification of skin diseases is that he did not conceive of an elementary lesion, *e.g.*, lichen, as a separate, individualized disorder. In this connection, Allbutt cites the dictum of Hebra: "We are at present in the A B C of dermatology and must learn to read the skin."[5] So the semeiology of the eye with reference to internal disorders has been interpreted by Schmidt-Rimpler or de Schweinitz. Furthermore the serial relation of diseases, like the differentiation of proteins with reference to their constituent amino-acids, applies not only to the individual but also to his relatives. When Charcot said that the patient is "only an accident in the history of the disease," he implied that the series may include the patient's ascendants, descendants and collateral sibs. There is merit in the English view that "we are killed not so much by diseases as by diathesis." Schön-

[5] For the semeiology of skin diseases in relation to internal disorders, see Sir J. Galloway, *Lancet*, London, 1921, I, 364–369.

lein's rheumatic purpura (peliosis rheumatica (1837), and Henoch's purpura with gastro-intestinal crises (1874) illustrate these correlations.

In 1888, Osler described an arthritic purpura, with gastro-intestinal crises, mucous hæmorrhages, albuminuria and sometimes fatal nephritis.[6] In 1895, he described eleven cases of polymorphous visceral lesions (gastric, renal, pulmonary) pivoting around the multiform erythemas (œdema, urticaria, purpura), with arthritis and other joint lesions. In a later paper (1900),[7] he stresses the futility of sharply separating angio-neurotic œdema from urticaria, voices the crying need for a "dermatological Linnæus" to straighten out the erythemas, and concludes that there is "a positive advantage in recognizing the affinities and the strong points of similarity in affections usually grouped as separate diseases."[8] In 1923–4,[9] Libman and Sacks described four cases of atypical verrucous endocarditis, involving all four valves, and showing such varied clinical phenomena as arthritis, mucous ulcers, pleuro-pneumonia, embolism, enlarged liver and spleen, glomerulonephritis, leukopenia, with erythematous and purpuric rashes, sometimes lupoid, sometimes of the nodular type found by Osler in subacute bacterial endocarditis. Again, the exanthematous fevers are specific infectious erythemas, with definite invasion and marked repercussion *via* the respiratory system, as well as renal, articular, cardiac, aural and nervous complications. The relations of herpes zoster with the specific infections, particularly varicella and encephalitis lethargica, are suggestive, as are the diathetic affinities of the visceral neuroses and their serial relations with the vagus and cutaneous nerves. In Allbutt's view, the rheumatic series includes purpura, urticaria, pemphigus, erythema, endocarditis, chorea, arthritis; the gouty series, dyspepsia, arthritis, phlebitis, arteritis, nephritis, angina pectoris, migraine, hypochondria, insanity, eczema, glycosuria, neuritis, bronchitis, haemorrhoids, haemophilia; the neurotic series (Charcot's neuropathic tree), insanity, chorea, eczema, pruritus, angina pectoris, gastralgia, asthma, sternutation, flatu-

[6] Osler: New York M. J., 1888, XLVIII, 675–677.

[7] Osler: Am. J. M. Sc., Phila., 1895, n. s., CX, 629–646.

[8] Osler: Brit. J. Dermatol., Lond., 1900, XII, 207–245. Osler was evidently unaware of Allbutt's paper of 1867.

[9] Libman and Sacks: Arch. Int. Med., Chicago, 1924, XXXIII, 701–737.

lence, catarrh, migraine and hysteria. He mentions a father and three children variously affected with cancer, lymphadenoma, pernicious anæmia and pseudo-hypertrophic paralysis. Where the serial relation exists or coexists in the same individual, the sympathetic-autonomic chain may be the locus of some of these transformations. The grouping of special families of bacteria as a finite series, with two extreme terms, *e.g.*, the paratyphoid-enteritidis or colon-typhoid group, the diphtheria group, the acid-fast group, fits in very well with the familial or serial aspect of diseases. The remote effects of syphilis and tuberculosis in family groups, *e.g.*, the nervous or endocrine manifestations, indicate the wide distribution of these major series in space and time. Hamer even regards the "catarrhal group of diseases," *viz.*, influenza, encephalitis lethargica, poliomyelitis and cerebro-spinal meningitis, as mere variants of the same virus, under special conditions of space and time. This is virtually Sydenham's view that a major infection may undergo a long periodic development over hundreds of years, with seasonal recurrences showing variations in semeiology of the most pronounced character. Changes in clinical manifestations of disease in space and time, including the decline in virulence of certain diseases like scarlatina, are susceptible of many explanations, the most plausible being Pasteur's doctrine of the change in virulence of microorganisms under different environmental conditions. Change in the relative susceptibility of people to disease is also of moment. The known antagonisms between certain diseases, *e.g.*, sarcoma *vs.* erysipelas or malarial fever *vs.* cancer, phthisis and neuro-syphilis are inexplicable but of great interest and therapeutic importance.

In the broader field of epidemiology, the varied and seemingly unrelated (protean) aspects of a major infection, as it crops out in different individuals in a family, camp, city, nation or continent, are the analogues of varying symptoms or groups of symptoms in the individual patient.

The prodromata of Spanish influenza in London, as interpreted by Crookshank,[10] were, in August, 1917, odd cases of acidosis, apparent tuberculous meningitis, cases resembling Brill's disease, tubercular peritonitis or scurvy; in January, 1918, pseudo-

[10] Crookshank: Proc. Roy. Soc. Med., Lond., 1920, XIII, Sect. Epidemiol., 170–172, 176–178.

typhoid cases (influenza of gastro-intestinal impact); in March–April, 1918, cases resembling encephalitis lethargica; in May, 1918, groups of virulent pneumonias recognized as influenzal by November. The major epidemic got in full swing about June–December, 1918, and during these months there were more cases than usual of Landry's paralysis, ascending and transverse myelitis, overlooked in the general furore about botulism and encephalitis lethargica. The vernal and autumnal prevalence of epidemic encephalitis and poliomyelitis respectively is also noteworthy. While not all these manifestations may have been influenzal, there were enough to demonstrate the protean aspects of the disease in different individuals under varying space-time relations. In Crookshank's reading, encephalitis lethargica is only one phase or member of the gigantic series "influenza," the early Latin records of which show unmistakable sequels of encephalitic type in the 15th, 16th, 17th and 18th centuries, comparable with the botulism, poliomyelitis, paralysis agitans, epidemic hiccough, etc., associated with the encephalitis of more recent date.

The doctrine of serial relationships in disease is of purely scientific interest and has little, as yet, to do with the actual practice of medicine. It implies, to start with, such a control of data concerning the familial and hereditary relations of diseases as was only possible to the family doctor of the past, and is now set off by recent books on the constitution, studies of notable families and the like. Its importance to the practitioner lies in the fact that our current "clinical entities" are merely fictitious adventures in conceptual shorthand, which, as Cnidians, we are compelled to use in practice as labor-saving devices, but which, like other phrases and symbols, should never deceive us as to ultimate reality. The following excerpts from Allbutt's three essays on classification[11] may be helpful:

"When we classify a disease, we do so, not to group together affections having a community of site or of aspect, nor for easy reference, nor for the symmetry of a page or volume, but to express thereby the greatest possible number of facts concerning its relations with other diseases."

[11] Allbutt: St. George's Hosp. Repts., Lond., 1867, II, 187–204. Brit. M. J., Lond., 1888, II, 285–292. Guy's Hosp. Gaz., Lond., 1906, n. s., XX, 448–455.

"False classifications do mischief, not only in asserting what is false, in declaring a kinship where no kinship exists, but also in drawing the mind away from the perception of real affinities. We are by them led to think of diseases as isolated disturbances in a healthy body, not as the phases of certain periods of bodily development."

"It is steadily forgotten that health is a diathesis as much as is scrofula or syphilis, and that each of these is a mode of growth. Some of these modes are more useful, others are less useful; but all tend, if unchecked, to pass through certain calculable cycles. That particular cycle which we call 'health' we prefer as being the most useful variety of the human plant, as it is the most varied and complex: for this reason we take it as a standard."

"An insight into what South called 'the fatal imposture and force of words' may be received from that profound reverence for names which we observe in children—including grown-up children—in contemporary savages, and in folklore. An inquiring child is dissatisfied until he is told the name of a thing; savage man still believes that to get at the name of a person is to gain a power over him. To know the name was to have the kind of power which was obtained in the effigy, the power vicariously to injure the person. Thus persons or gods would take false names in order that the real name might be sheltered from malevolence, or that the worshipper might be spared the fulmination of the name."

"In mediæval times so fastidious were logic and abstraction that practice became a vulgarity, and he was the greatest teacher who carried his pupils farthest from things."

"Unfortunately we go on building with names when the things are altered or wasted away, as sometimes beavers pathetically persist in constructing dams and canals when the water has gradually dried up, or has changed its course. If we realize how a word may survive to oppress and mislead us, as other ghosts do, when the underlying thing has dissolved, we shall be more careful in setting up imposing names, as we physicians are very prone to do, until we are sure that the thing is there; and in no case shall we let a name give an absolute value to temporary or developing notions."

"Some of you who have heard my teaching before must forgive me if I repeat my insistence that the name of a disease is not, as it is continually regarded, a thing. There is no such thing as typhoid fever, as angina pectoris, as spleno-medullary leukaemia, and so forth; the things so called are Wilkinson, Johnson, and Thompson, who after their kinds are afflicted not alike, but within such limits of similarity as to lead us to class them together and to form a general conception of them. And yet still we overhear at learned societies physicians, whose shoe-latchets I am not worthy to unloose, contending, even with heat, whether this name or that is a 'morbid entity.'"

"If we are to speak of 'entities' in disease, these must be not the names, nor even our concepts, but the things—the thing Thompson and the thing Wilkinson in certain phases of their being. The moment we depart from these objects we desert the names of things for the names of abstractions in which no entity can lie, the name being but a label to denote a somewhat arbitrary and ideal group of characters never perhaps manifested in nature as a whole, but to which certain patients are continually approximating. I do not pretend for a moment that we can do without abstractions; without them reasoning would be impossible: our safety lies not in avoiding reason but in being quick to recognize the tendency of ratiocination to carry us away from the only pregnant subjects of reason—namely, from things, with which reason should habitually and steadily concern itself."

"Diseases are not even species, such as cats and toads, but abnormal, though not altogether irregular behaviors of animals and plants. Once more; not content with this ambiguity we entertain the converse notion also that, give it a name big enough, we can make a disease of a symptom. For example, bradycardia and tachycardia, symptoms, in their degrees, of many a morbid process, impose upon the unwary student as themselves 'morbid entities,' a notion twice removed from the truth."

"The pathologist has seduced us into allowing him to use the label of arteriosclerosis as the name of a disease. Now, if for the physician a disease is a series of symptoms recurring with such uniformity that we think it convenient to distinguish it with a name, arteriosclerosis is not such a series; it is not a series of symptoms at all; it is a result, a statical result, of foregone

symptoms, probably of more than one series, and, if so, probably itself a compound name even in pathology. The physician who recognizes the imperative duty of dwelling in things ought to guard himself from being supposed to mean only things that stand still; his sphere is, on the contrary, with things in motion—he is a master of dynamics."

"Mr. Galton, Dr. Isambard Owen, and Dr. Billings tell me that when we desire something more than the notes of the family history of the same disease, when he ask for a scheme of all the diseases, fatal and non-fatal, found in one family, we desire that which does not exist. The only persons indeed who could make adequate records of this kind are family medical advisers who have watched over the same households for long periods of time. Public opinion is not yet alive to the importance of such records, and as busy doctors decline in years, they are less willing to embody the invaluable results of their experience. Thus a synopsis of all the maladies current in one family tree is rarely or never met with; although we admit that the individual is but one link in a chain."

"Much of the work which is done in our laboratories and dignified, not improperly perhaps, with the title of research, much plotting of curves, much watching of levers and thermometers, nay, not a little morphological dissection and cabinet making, are really little more than clerk's work. To be no more learned in facts and opinions, with whatsoever skill in methods, is not to make knowledge. Bundles and files of facts are not science until the man with the formative, let me say the creative, insight comes along, who by the fusion of intellect and imagination seizes upon the significant facts, which give him the lines on which to build up these aggregates of materials into a conceptual edifice. As we abstract we depart from things; and the wider the successive abstractions, the farther we are leaving things behind, the more and more are we eliminating those contingencies in which processes, organic and inorganic, essentially consist."

"The laboratory, invaluable, essential as its work is generally speaking, is apt to dwell only half in things; and nature will have no half loves. It is in the full commerce of nature and men that the problems fruitful of science arise; their events and processes, vulgar as they may be, are full of the unexpected, and

it is beyond the imagination of any scientist even to conceive such combinations and emergencies unless he is conversant with things. Furthermore, if he tried never so generously to imitate natural processes and events on his artificial platform, he could not possibly reproduce them in their complexities. The navigator of models in a tank would cut a poor figure as captain of a man-of-war in the Atlantic. Thus, without periodical steeping in the world of practice, theoretical researches tend to remoteness and vacuity. It is by continual touch with Mother Nature that we renew our strength, and for her nothing is common or unclean."

"Day by day we see well-meaning men confusing themselves and others by ignoring that in science law is not a rule imposed from without, but an expression of an intrinsic process. The laws of the lawgiver are impotent beside the laws of human nature, as to his disillusion many a lawgiver has discovered."

"To find new arms against disease, we must have a truer conception than we mostly have of the genesis of disease. No mere enumeration of symptoms or of pathological detail will suffice for this, nor even a full description of diseases severally. We have to work out the genetic affinities of diseases, their origins, parent stems, and alliances, as well as their issues. We can have no complete therapeutics until the science of comparative nosology is in great measure constructed—a science as yet scarcely begun—nay, as yet scarcely recognized."

"Medicine likewise, because it deals with things, has always been for our serener circles, a Cinderella, blooming maid as happily she has grown nevertheless."

The conclusion of the whole matter is implicit in Osler's dictum—"Treat the patient rather than the disease"—and, for neurology particularly, in the line of Weir Mitchell:

" 'Tis not the body but the man is ill."

F. H. Garrison

OBSERVATIONS DURING THE PERIOD OF UNDERNUTRITION IN GERMANY

FRIEDRICH VON MÜLLER, Munich

(Delivered before The New York Academy of Medicine, October 21st, 1926)

I appreciate most deeply the kind invitation of The New York Academy of Medicine and I did not hesitate a moment in accepting it because I wish to do my best in promoting good understanding between the medical men of the United States and of Germany.

I propose to review the years of undernutrition suffered by the German people during the times of the blockade and revolution and to look on this period not from a political but from a medical standpoint as one would view a great experiment from which we may try to draw some conclusions.

From the very beginning of the war we realized that the available food supply would not be sufficient for the German population because statistics had shown that during the years of peace it was necessary to import many million tons of food for man and cattle and large amounts of fertilizers for the fields. How could the country stand a period of total blockade like a fortress surrounded by enemies on all sides? We therefore were not astonished when we realized early in the Fall of 1914 that the bread supply was insufficient. It was possible to meet this difficulty in two ways. The first was a legally enforced distribution of the entire bread supply to the whole population by means of the bread cards. The second method was a greater utilization of the wheat taken for bread.

The first of these two ways proved beneficial and it was soon necessary to extend the card system with fixed prices to the distribution of meat, milk, eggs, fat, potatoes, and finally to nearly every food-stuff. One may regard a legal distribution of food as a socialistic method but it is necessary to prevent the rich from leading an opulent life while the poor starve as they really did in Constantinople and Vienna. Of course such an enforced restriction and distribution is possible only in a state with strong government and good civil order. With the breakdown of the social order during the revolution of 1918 the regular food supply collapsed and a wild illegal struggle for food began.

The second method of regulation, coarser milling, was of doubtful value. In times of peace the rye and wheat were normally milled so that 60 per cent. was utilized for bread. With the new law it became obligatory to use 95 per cent. so that the bread contained all the bran and this was not digestible for man. This same bran, however, is digestible for the cow and the cow can produce milk from it. Milk almost disappeared from the cities and could only be supplied to infants and sick people. Bread became worse and worse, especially in 1916 when potatoes and turnips were mixed with the whole wheat. The question of the bread was all the more important because, on account of the lack of animal food, bread supplied almost two-thirds of the necessary food calories.

Of all the restrictions the deficiency of *meat* was the least important and produced no bad results. Hyperloyal physicians and men like Ragnar, Berg and Hindhede insisted emphatically that a vegetarian diet would be of the greatest benefit to the population, but more critical observers were not able to see the promised benefits from the lack of meat—with one exception. *Gout* disappeared entirely and in fully developed cases of gout the affected joints showed distinct improvement. Among the population in general the amount of uric acid in urine and blood fell to half of the normal level, not lower. It reached the level of the endogenous uric acid.

Is it not curious that the human body even in restricted diet is able to build up the complicated nucleinic substances while it is absolutely unable to destroy uric acid? Not all of the chemical reactions in the body are reversible.

After the experience of the war there is no more doubt that the development of gouty affections (I do not say the origin of gouty *constitution*), is to a great part dependent on rich food and alcoholic drinks.

The triumph of the vegetarian apostles like Hindhede did not last very long. Soon after the war the whole population forgot the benefactions of the vegetarian diet and eagerly went back to meat—and two years afterward some cases of gout reappeared.

With few exceptions the restriction of diet did not show really bad effects until the Fall of 1916. We all became thinner and

the average weight diminished about 12 per cent. Stout people and heavy eaters lost far more than people of normal shape and it was not rare for the obese to lose 70 to 90 pounds. Real obesity was no more to be seen on the streets and the experience of this period of undernutrition has clearly shown that common obesity is not so much caused by endocrine and constitutional disorders but to a simple discrepancy between food intake and expenditure. The basal metabolism of obese people is not lower than in the individuals of normal shape, but very often a little higher.

Things went on very tolerably until the Fall of 1916. About this period milk disappeared almost completely, cheese, butter and eggs became scarce, bread hardly more deserved its name and worst of all the harvest of potatoes in this year was very bad. Instead of potatoes we were obliged to eat indigestible turnips for breakfast, lunch and supper. The tragedy began. Previously the loss of weight had been due to a diminution of the fat stores but now appeared a wasting of the muscles, and with it came a loss of strength. Exertion grew more and more difficult. In my trips to the numerous hospitals in the field and at home I found it difficult to climb the staircase of railway stations. In the factories they needed three men to accomplish the same work formerly performed by two. Even the children lost the desire to play. In the civil prisons the criminals became so weak that they could no longer work and in the insane asylums patients who formerly were excited remained quiet and stayed all day long in bed since this was the only place they could be warm. The body-temperature was below 96.8° F. and the pulse rate sank from 50 to 40. Even among the population in general, the systolic blood pressure fell to 100 and lower. In addition to the bodily emaciation came a mental exhaustion, depression and irritability, and a large part of the subsequent moral breakdown was due to this state of mind. There was forced upon our notice a mysterious increase in the death rate, particularly among old people. Almshouses and asylums were slowly emptied by the increased mortality and in these houses we first saw the cases of edema. This edematous swelling of face, limbs and body slowly manifested itself among the free populations of the cities also.

In order to explain these problems, experiments were made in many places including our own clinic. Dr. Jansen and I made some observations on the inmates of asylums and prisons and also on some students of medicine in our clinic. All of these persons were put on the diet as prescribed by the legal distribution. First it was necessary to find out the real caloric value of the ration since it would be wrong to estimate its value from the analytical tables used in times of peace. We collected and dried the food for the whole day and analyzed a sample by combustion in a Berthelot bomb. From the caloric value of the food it was necessary to subtract the caloric value of the feces. This examination made it evident that the real caloric value of the diet was not 1,350 calories as officially given, but only 1,100 to 960 calories on account of the large proportion of unabsorbable material. A nutritive value of 1,100 to 960 calories is of course far less than is necessary to maintain the needs of the body. It is even lower than the resting basal metabolism while for light work 2,300 calories at least are necessary. But our subjects were *obliged* to perform some work and muscular work cannot be produced more economically than its real caloric value. And in addition we had to work harder than during the times of peace. It was therefore evident that people on this diet must lose weight and body substance. Examination of the nitrogen content of the *food* showed that the amount of protein in the diet was sufficient and even much higher than the nitrogen minimum of 3 to 4 grams a day, but the nitrogen *excretion* by urine and feces was found even higher than the intake since the sparing effect of the carbohydrates and fats were insufficient.

I cannot understand how Johansson in his latest article could deny the sparing effect of carbohydrates and fats upon the protein metabolism. Did he not realize that this sparing effect is only manifest in cases of *deficiency* and not in cases where carbohydrate is added to a diet that is already sufficient? The negative nitrogen balance in our cases was therefore caused by a deficiency of fat and carbohydrate in the food and as soon as we gave our subjects additional fat or sugar, the nitrogen equilibrium was reestablished and there was even some nitrogen retention.

A determination of the basal metabolism by Zuntz and Loewy and by ourselves made it evident that the basal metabolism was

distinctly diminished in comparison with the metabolism in time of peace and even reduced in a higher degree than the loss of body weight. While the body weight was diminished on the average 10 per cent. the diminution of basal metabolism amounted to from 12 to 15 per cent. These results agree with those of Benedict obtained with his students in the condition of prolonged undernutrition. It was evident that an economical *adaptation* had taken place at a lower nutritive level. In consequence there was a reduction of body temperature, pulse rate and blood pressure and the vitality of other organs with a resulting diminution of mental and bodily efficiency and a diminished resistance against disease. Tuberculosis ran a more malignant course and a frightfully increased mortality,—about 50 to 90%. Pneumonia and other infectious diseases and surgical operations showed a diminished tendency towards recovery.

During the early part of the war some optimistic physicians had published the opinion that the German people had been overfed and that a reduction of food was not only allowable but also beneficial. But when the real diminution in food took place it became clear that it was not a benefit but a menace.

This diminution of the basal metabolism during prolonged undernutrition finds its antithesis in the *increased* metabolism during periods of overabundant feeding. Grafe uses the expression of "Luxuskonsumption," a conception stoutly denied by Karl Voit, Rubner and my former assistant Lauter. It is evident that every increase in the oxidative processes which is not used for muscular work must find its expression in an increased production of heat (I do not say in an increased body temperature). In fact a well-nourished man can bear cold temperatures much easier than a starving man who can scarcely warm himself while in bed, and an overnourished man or animal may even find difficulty in getting rid of his overproduction of heat. But is it really a luxury when the state of nutrition of the body is improved (naturally only to a certain degree) and as a result the resistance against certain diseases is increased? Thus it was possible for me many years ago to show that during the recovery from severe infectious diseases like typhoid fever the basal metabolism was distinctly raised in accordance with the increased

appetite of the patients, and we employ abundant feeding for the curing of tuberculosis. The question of the increased basal metabolism in overnutrition stands in close connection with the specific dynamic action of Rubner and the law of Lavoisier's that the metabolism is increased after the taking of food. The specific dynamic action is only a special case of the general law that food raises and starvation diminishes the production of energy. Rubner has shown that this production of extra energy after taking food cannot be used for the production of muscular work and therefore we may conclude that it does not take place in the muscles but somewhere else, possibly in the liver. Among the different food-stuffs protein produces the most energeric rise in heat production. These are the facts and the only question is how we shall explain the phenomena.

There are three possible ways to explain them. The first and simplest method of explanation is that the human body behaves like a stove and that the more fuel you put in the more is burned, and in consequence the more heat is produced.

The second theory is that food and especially protein act as a stimulus, and Graham Lusk has shown that especially some amino-acids like glycocoll and alanin increase heat production while other amino-acids do not. According to Lusk's work the amino-acids which produce extra heat are nearly the same as those which can be transformed into sugar.

The third way of explanation is that the increase of heat production is due to the work of the intermediary chemical transformations. Thus the transformation of sugar into fat or protein into sugar needs energy and thereby heat may be set free. Included in the work of this intermediary metabolism is the production of hydrochloric acid in the stomach and the work of all the other digestive glands, and I think that they need a lot of energy. I am inclined to propose this last theory. The specific dynamic action is not constant but it is variable and changes not only with the different kinds of food-stuffs but also with the general state of nutrition and especially in connection with the glycogen content of the liver.

The question of overfeeding has been the subject of excellent work by Lusk, who was able to show that in hogs, during a tre-

mendous overfeeding with carbohydrates, a considerable increase of oxidation takes place, the metabolism being increased 100 per cent. above the former basal metabolism, this increase lasting not only twenty-four hours but also during the whole period of overfeeding. But the greater part of the carbohydrate in the food was transformed into fat and deposited in this form in the tissues. The respiratory quotient was increased up to 1.5, although it is only 1.0 during combustion of fat.

The great excess of oxygen output over the intake can only be explained by the transformation of a great amount of carbohydrate into fat, since the molecule of carbohydrate is far more rich in oxygen than that of fat. The enormous increase in the oxidative process may be explained by the assumption that the transformation of carbohydrate into fat requires energy, and the production of this energy proceeds with increased heat production. I therefore believe that the increase in heat production by constant overfeeding can well be compared with the temporary increase of metabolism, caused by the specific dynamic action of foods and that both processes can be explained by an increase in the intermediate chemical work required.

The fourth possibility is put forward by Johansson of Stockholm. A few months ago he published some new ideas about the metabolic processes,—some ideas which may change or alter our previous conceptions. Upon the basis of the researches of Meyerhof and Hill, Johansson has established a theory that the consumption of energy does not take place during the active work of muscles and glands but during the period of *recuperation*—"first the work, secondarily the consumption of energy."

May I make a comparison? Energy is used not in firing the gun but when it is reloaded; not in shooting an arrow, but in drawing the bow. Hill has indeed shown in his splendid experiments that energy consumption, measured in terms of heat exchange, takes place not only during the contraction of the muscle but also—although to a lesser degree—for a certain time afterwards during the period of recovery, and he has shown that this period of recovery is accompanied by an increased consumption of oxygen, which is to say by oxidation.

But I do not believe that it is permissible to draw from this experiment the conclusion that the whole consumption of energy and the whole production of heat fall in fact in the period of recovery, as Johansson wishes to say.

Hill accepts the view of Meyerhof in proposing the theory that during the period of work the glycogen of the muscles is converted into lactic acid and that during the period of recovery the lactic acid is converted back into glycogen. This endothermic reconversion of lactic acid into glycogen requires energy and takes place with consumption of oxygen, but the process takes place without the entrance of oxygen into the molecule, since the relationship between oxygen, carbon, and hydrogen is the same in lactic acid as in glycogen.

The consumption of oxygen and energy must therefore be the consequence of oxidation of other substances. Meyerhof and Hill assume that a part of the lactic acid, either one-fifth or one-fourth, is burned to carbonic acid and water, to provide the energy for the resynthesis of the glycogen, and recently Myerhof has stated that this energy may be produced also by oxidation of fat.

Indeed Zuntz has shown that during a state of glycogen deficiency, work can be produced by oxidation of fat alone, and Lusk has shown in brilliant experiments that in this condition the respiratory quotient is so low as to indicate that the energy production takes place from combustion of fat. Moreover Krogh has shown that the production of energy takes place in a more economical way when carbohydrate is burned than when fat is consumed.

Meyerhof, Hill, and Johansson assume that during work glycogen is transformed into lactic acid, and that during the period of recovery this lactic acid is reconverted into glycogen, only one-fifth or one-fourth of the lactic acid being burned. Within this theory is hidden a little of perpetual motion, and when we assume perpetual motion we are always wrong.

Our acceptance of this theory meets with some difficulties.

First, the oxidation of lactic acid is not so easy as that of the aldehydes, such as methyl glyoxal, glycerol aldehyde, or acedaldehyde.

Secondly, according to this theory we must conclude that an enormous amount of lactic acid is produced in the muscles during hard work. Lusk has calculated that during hard work a man may liberate not less than half a kilo of lactic acid per hour, and in seven hours of hard work might liberate the enormous quantity of 3.5 kilos of lactic acid. Of this amount four-fifths would be resynthesized into glycogen.

Third, Hill himself states that "in a muscle kept in oxygen, no lactic acid ever appears." Lactic acid appears in muscle preparations only when contractions occur in an atmosphere free from oxygen. Lactic acid is therefore found only in the absence of oxygen.

I believe, therefore, that the older assumptions are not yet disproved. The older theory was that the energy of muscular work is produced by direct combustion of glycogen and sugar, with the intermediate products of easily combustible materials such as pyruvic acid, methyl glyoxal, and acetic aldehyde, and that this energy can also be produced by the combustion of fat.

I do not care much which of these theories is right. The facts are established and the experiences during the period of undernutrition have clearly shown that our views about the energy metabolism are right and that they fully agree with the law of the conservation of energy.

After consideration of the *quantitative* metabolism let us turn to the *qualitative* and *pathological* changes seen in men during the period of undernutrition. The most important symptom was the hunger edema with its pale, swollen and mask-like face. One could believe that this pale color was due to a severe degree of anemia, and examination of the blood showed that the red blood cell count was somewhat low but this apparent diminution of the red blood corpuscles was due to a hydremic condition and not to a severe reduction of the red blood cells. The blood serum was indeed very rich in water and the protein content was extremely low, even down to half of the normal rate. The amounts of calcium and lipoid bodies were distinctly lowered. The sugar content varied between very low and nearly normal. On the other hand the sodium chloride was extremely high, even up to 0.8 per cent. We can explain this phenomenon by Donnan's law which states that the content of salts must rise in body fluids when there is a diminution in the protein and other colloid materials. This is necessary in order to maintain the normal osmotic equilibrium. The body tissues as well as the blood serum were overfilled by a fluid which approached the composition of normal physiological salt solution. We can therefore understand the abnormal craving of the patients for salt in the food. With this

the excretion of sodium chloride in the urine increased to as much as 40 grams a day and more. It was evident that the function of the kidney was normal and that the edema was not due to an insufficiency of the renal function but was of extra-renal origin. We may ask if it was the low content of protein in the blood serum which caused the transudation of salt solution into the tissues or if it was the diminution of the calcium and especially the lipoid substances which led to an abnormal permeability of the walls of the blood vessels.

It was interesting to see that the content of hemoglobin in the red blood corpuscles was not diminished but was increased and the color index rose far above 1 and even as high as 1.9. In stained blood smears the red corpuscles showed an appearance similar to that found in pernicious anemia. But this high color index had nothing to do with pernicious anemia and it is evident that we may conclude from this that the high color index is not the essential characteristic of pernicious anemia, but that in this disease other factors are of greater importance.

We have seen that even in severe cases of hunger edema no real anemia was present and as a matter of fact during this period we saw no cases of chlorosis. We can conclude that even during a time of serious lack of food the synthesis of the complicated hemoglobin molecule takes place without difficulty. Apparently living organisms from the yeast cells up through the whole series of animals find no difficulty in producing the complicated nitrogen-containing ring of the substituted pyrrol molecule and from simple open chains combine the coloring matters of porphyrine, chlorophyl and hemoglobin. How extraordinary that the body is not able to break down the once established hematine farther than bile pigment! Therefore here as in the case of the purine metabolism we have an irreversible reaction. My former assistant, Prof. Hans Fischer, in the last few months has had the good fortune to be able to prepare synthetically hematoporphyrine and he pointed out that the production of hemoglobin by animals and man by far exceeds in amount the artificial production of all the paints and dyes of industry.

During the second half of 1917 hunger edema disappeared in a relatively short time. This was the result of a good harvest of

potatoes and these potatoes allowed us to give once more sufficient calories. Potatoes indeed saved the German population from the extremes of starvation.

When a patient with the hunger edema really began to improve there was a tremendous increase in the output of urine. With this came an excretion of as much as 160 grams of sodium chloride in a few days and a rapid loss of weight. In cases where we were able to give abundant food again we saw to our astonishment that the patient did not increase in weight. But we must not forget that he had to replace the water in his tissues with fat and that a kilo of water is as heavy as a kilo of fat and that the same volume of fat tissue is not nearly as heavy as the same volume of salt solution. Therefore the weight of a person is a most uncertain indicator of his state of nutrition.

When we saw the first cases of edema we believed that it was a manifestation of avitaminosis. However the well-established types of avitaminosis did not appear. We never saw diseases like beri-beri or scurvy because we had plenty of potatoes, turnips and other vegetables. But in Asia a part of the English army surrounded by Turkish troops and cut off from all fresh food suffered from a severe outbreak of scurvy.

Are we really to consider hunger edema as a symptom of avitaminosis? This makes it necessary to define what we mean by vitamines.

The characteristic signs of avitaminosis are:—

1. A diminution of fertility.
2. A retardation of development and especially of the growth of young animals and children.
3. Symptoms of malnutrition and disease in the fully developed adults.

Diminished fertility was indeed one of the characteristic symptoms during the period of inanition. The official register showed that the birth rate had diminished enormously, falling to half the figure of the rate before the war. Abortions became frequent, in many young women menstruation ceased and in men there was a marked diminution in the libido. We can see therefore that there was a distinct diminution in the function of the gonads. Zahn has shown by his official statistics that during the war the

diminution in the number of births amounted to the same low figure as if for one whole year there had been no births in Germany.

The worst part of this whole affair was that we looked upon this not as a calamity but as a blessing because we were sure that we would not have either enough food or enough work to maintain them. It is a regulation of nature that in times of want the number of individuals decreases.

But one question. Is the diminution of fertility a characteristic and pathognomonic sign of avitaminosis? I think not.

Professor Stieve in Halle has shown in his experiments on mice, rats and geese that not only inanition but more so overfeeding and obesity checks fertility in both male and female, and brings about severe atrophy of the gonads. Furthermore he could demonstrate that fear, confinement and other severe *mental* influences damaged materially the power of fertility and produced an atrophy of the gonads as demonstrated by the microscope. Therefore we may conclude that the diminution of the birth rate is not a characteristic sign of avitaminosis.

Next we shall consider our second point, the development of children. During the war the increase in weight of children fell off markedly, in Munich 4 per cent., in some other cities as much as 12 per cent. The growth in height was only slightly diminished, ¾ of one per cent. on the average and according to some reports not at all affected.

There was indeed some extension of rickets among the children, especially in Vienna and some other large cities, and during the years of adolescence coxa vara with its characteristic pain became frequent. Older people and hunchbacks complained of pains in the back, X-rays showed slight osteomalacia and kyphosis became worse. Treatment with phosphorus and cod-liver oil brought distinct improvement.

The "Wachstumstrieb," the *impulse* for growth therefore was *not* diminished. The observation of the fact that the affections of the bones improved distinctly after the administration of cod-liver oil could be explained as a symptom of avitaminosis due to deficiency of vitamine A. We must remember that vitamine A is not equivalent to "Wachtumsreiz," the stimulant of growth, but

that this vitamine A is a necessary complement to bring about growth. When there is no stimulus, vitamine A cannot produce growth but growth can be checked by a deficiency of vitamine. The mysterious "Wachstumstrieb," the stimulus for growth is more dependent on the glands of internal secretion like hypophysis, thymus and thyroid gland but it can only act with the help of the vitamines.

Among the vitamines which are necessary for growth vitamine A seems to be the most important. A. F. Hess has shown, and some German investigators have corroborated, that vitamine A does not belong to the phosphatids like lecithin but that it is related to cholesterole and arises from this substance under the influence of light. Windhaus has established the chemical formula of cholesterine. The alcoholic group is the place where it can combine with fatty acids to form esters. We may assume that the double binding is the place where light acts and produces some oxidation. This vitamine is therefore no longer a mysterious body and this helps us perhaps to have a better understanding of the nature of the vitamines in general.

Would it not be possible to characterize the vitamines as all those substances which are necessary for growth and maintenance of health which cannot be produced by the organism itself but must be supplied from outside? I should like to support the old view of Liebig that the soil needs at least a certain minimum of every single substance necessary for the growth of the plant. In the same way it holds true in the animal body that growth is only possible with a minimum of iodine and Halsted has shown that growth is checked when there is no iodine in the food. I therefore conclude that iodine is a vitamine, like calcium, magnesium and iron. Of course growth is not possible without protein but it is not protein itself which is necessary but the single amino-acids. It has been proved that growth is impossible without lysine and the establishment of nitrogen equilibrium and the prevention of death is possible only with a mixture of amino-acids which contains cystine and tryptophane.

Now we come to the question as to whether cholesterole can be formed in the body or if it must be given in the food. Thannhauser has shown in his experiments that the excretion of choles-

terine in urine and feces is nearly the same as the intake in the food. But should we not include in this balance between intake and output the bile acids also, the chemical formula of which is so closely related to that of cholesterole, that we must conclude a common origin? The production of bile acids for twenty-four hours amounts in the dog to one or two grams while the cholesterole in the food is not more than a few milligrams. We may therefore conclude that the complicated formula of cholesterole and bile acids may be combined in the body and Windhaus believes they are built up from the straight chain of oleinic acid.

Pirquet and other authors have assumed that the phosphorus-containing lipoids like lecithin are not among the vitamines, but that they can be produced by the body itself. I am not perfectly sure of it. Why then does the yolk of the hen's egg and of all other eggs contain so large an amount of phosphoric lipoids? It seems to me that they are necessary for the building up of the embryo. On the other hand it is of interest that in the egg neither pyrrol nor purine bodies are present although during the development of the chick within the shell a great amount of blood pigment and nucleinic substances are formed. It is evident that pyrrol and purines do not belong to the vitamine group.

With the end of the war we hoped that the blockade would end and that food supplies would come in again to the starved population. But the blockade continued. The deprivation of food became extreme, the mental state of the population broke down, all bonds of order collapsed and the revolution broke out, with the slogan of "more food, at lower prices, and shorter hours of work." In this worst period of our country the work of charity from America set in and we never will forget our thanks for this help of charity given to the starving German population, by our American friends, especially by the Quakers and last, but not least, by the American physicians.

Gentlemen: I have tried to draw a sad picture of the starvation which the German population suffered during the war. War is bad, it brings enormous sufferings and losses of life and happiness, it disturbs the mind and evokes cruelty. But war is able to bring forward the greatest mobilization of energy, self-denial and sacrifice.

Revolution is worse, it loosens all bonds of authority and releases the lowest instincts in the worst classes of the population. The gain is small and the loss of culture great. The recovery from the false ideas is slow, very slow.

I sincerely hope that your beautiful, rich and highly cultivated country will never undergo the horror of revolution which is the most cruel of all kinds of war.

Abstracts of papers presented before the Section of Genito-Urinary Surgery, March 17th and May 21st, 1926

(a) URIC ACID AND URATIC STONES IN THE KIDNEY; URIC ACID SHOWERS AND DIAGNOSIS

EDWIN BEER

One of the most serious pitfalls in diagnosis encountered by surgeons, internists and radiologists is in the recognition of uric acid stones and the clinical pictures induced by such stones or by showers of uric acid crystals. The clinical pictures presented by the above conditions have interested us for a number of years. Originally, it was recognized that stones of this composition caused no shadow on the X-ray plate, and the report of a negative radiogram was recognized as of but little value. With the introduction of the Bucky diaphragm these uratic stones in the bladder were more regularly caught on the film though the shadow, at best, was only faint. In the upper urinary tract, the analysis of stones passed showed that in this vicinity about eleven (11%) per cent. of the stones were composed of uratic salts and uric acid. These the X-ray regularly failed to show despite the use of the Bucky diaphragm.

In a series of six cases presented at this meeting, it was shown that the diagnosis of the presence of uric acid calculi in the kidney pelvis could be made by combining pyelography and the passage of wax bougies. These stones in the kidney pelvis at times attained large size, and in the pyelogram showed their presence by a well-marked filling defect. Very frequently in

these cases, the wax bougie will obtain a positive scratch-mark. It is possible that many patients who have more or less typical kidney colics, whose X-rays are negative, may harbor such translucent uric acid stones in their kidneys and, unless they are carefully and repeatedly studied, the diagnosis of the exact condition may remain obscured.

Closely related to this group of patients with sizable calculi in the kidney, there is a still larger group of patients who suffer from typical colics caused by the passage of showers of uric acid crystals. The voided specimen when fresh rarely shows the presence of these crystalline substances but on standing, with the separation of the colloidal nebecula, crystals drop out. This phenomenon is present in many cases that never complain of urinary symptoms, but when it is present in patients with typical unilateral or bilateral kidney colics and when, moreover, diminution in protein intake is followed by a relief of all symptoms and the disappearance of the crystals from the standing urine, it is only fair to assume a definite relationship between complaint and appearance of uric acid crystals. What probably happens in these cases is a precipitation of crystals in the upper tract and as the colloidal solubility is re-established (reversible colloid), the uric acid goes into solution again so that in the voided specimen there is no evidence, as a rule, of the presence of these crystals.

How often these cases are misinterpreted—perhaps interpreted as kinks of the ureter, strictures of the ureter, perinephridies, and the like—it is difficult to say.

(b) THE CURVE OF 'PHTHALEIN EXCRETION; ITS INTERPRETATION AND CLINICAL USE

A. J. Crowell
Charlotte, N. C.

The dye test for kidney function as usually employed does not tell the entire story. While the 'phthalein output and blood nitrogen are the usual renal function tests used as guides to operability of prostatic cases, there have been observed distinct

discrepancies between the information obtained by these methods and eventual clinical outcome.

Shaw has pointed out some characteristics of the plotted curve of dye excretion which give additional information of great value in choosing the time of operation and in offering a prognosis. Repeated clinical test of this method in 100 cases has confirmed his findings and convinced us of its usefulness.

Characteristics of the type curves, with actual case curves.

Case reports of simple and complicated prostatic cases with repeated dye curves which aid in guiding the management of these cases.

(c) THE ROLE OF A SPECIFIC BACTERIUM IN THE PRODUCTION OF URINARY CALCULI

B. H. Hager and T. B. Magath

In a previous publication attention was called to a specific bacterium as the etiologic factor in production of incrusted cystitis with alkalin urine, and at the same time mention was made of the fact that free, floating stones of the bladder were occasionally observed in laboratory animals in which incrusted cystitis was experimentally produced. This particular species of bacteria had not previously been described, and it was placed in the genus Salmonella.

Twenty strains have been studied, and as a result it is believed that the organism does not belong in the genus Salmonella, because the majority of the strains liquefy gelatin, grow with ameboid colonies, and all appear to be protein destroyers. They have the characteristics of the genus Proteus except that they do not ferment sucrose. Since the fermentation of sucrose appears in the generic description of Proteus, it was proposed to modify the genus to include some forms that do not ferment sucrose. As this seems to be the most logical disposition of the species, the name becomes *Proteus ammoniae*, there being no other species corresponding to this.

From experimental work and a study of the cases of incrusted cystitis associated with alkalin urine, it seems that the presence

of Proteus organisms in the bladder is important. Whether any other member of the genus beside *Proteus ammoniae* can produce the pathologic picture made by this specific organism is not known. The facts are these: this organism was isolated in many cases of incrusted cystitis, and in all cases so clinically diagnosed since the identification was first made. No other organism of this genus was isolated in such cases, but other members of the genus Proteus, in cases other than incrusted cystitis, were occasionally isolated.

Because of the occurrence of true vesical calculi associated with alkalin urine and because of noting free, floating stones in the bladders of animals in which incrusted cystitis was produced, the authors sought to determine whether or not there existed any relationship between vesical calculi, found in alkalin urine, and the presence of *Proteus ammoniae*.

Four case histories of vesical calculi with alkalin urine from which *Proteus ammoniae* were isolated are presented.

(d) EXPERIMENTAL AND CLINICAL PROOF OF THE VALUE OF INTRAVENOUS GERMICIDAL INJECTIONS IN LOCAL AND GENERAL INFECTIONS

H. H. Young, Baltimore
J. H. Hill, Baltimore

Dr. Young traced briefly the history of intravenous therapy beginning with the first injections made by Sir Christopher Wrenn, the great architect of St. Paul's Cathedral in London. He showed that numerous great names in medical history have been associated with attempts to treat infectious diseases by intravenous drugs, culminating in Ehrlich's presentation of his salvarsan and claims of a therapia sterilisans magna in spirochetoses and spirilloses. Dr. Young gave in detail the work at the Brady Urological Institute in an effort to obtain a more effective, less toxic, less-irritating germicide, which culminated in the discovery of mercurochrome, which could be introduced into animals

and human beings intravenously in doses of 5 mg./kg. of body weight with impunity. The first work which had shown this drug to be of value in the treatment of wounds and inflammations of mucous surfaces had been extended to the intravenous treatment of general infections, septicemias, infectious diseases and local infections. He cited at first almost moribund cases in which septicemia was rapidly cured by intravenous injections of mercurochrome, described hundreds of other cases and infections of various types in which the drug had been employed. He presented detailed tabulations of over 600 cases with many extraordinary results in diseases which have heretofore been always fatal. In 173 cases of septicemia, 109 (63 per cent.) were cured. He cited 200 cases of pneumonia from the clinic of Freeman and Hoppe in which the mortality had been reduced from 35 to 10 per cent. He also showed tabulations of various other local and general injections—erysipelas, cellulitis, furunculosis, arthritis, pyelitis and various infectious diseases in which marked benefit was obtained by intravenous mercurochrome therapy. A summary of 611 cases of various types showed that 74 per cent. were cured or immediately improved. He quoted additional series from the clinic of Dr. Du Bose in which 100 cases were treated with only 5 deaths. An analysis made of urinary findings before and after mercurochrome showed that in 510 cases in which accurate information was at hand, only 11 showed more than transitory albuminuria or casts and 10 of these 11 cases were practically moribund and died within a few days, in spite of mercurochrome infections. He quoted statistics from the department of pathology of the Johns Hopkins Hospital and showed conclusively that there were no ulterior effects produced by mercurochrome upon the kidneys. As to dosage, he admitted that he frequently used large doses which had been followed with very severe reactions. Large doses were probably of importance in fulminating and desperate cases such as severe septicemias, but in milder cases he believes that now one should begin with 1 to 2 mg./kg. of body weight and by repeating the injection every two to three days, get equally good results with almost no reaction. The final conclusion was that in mercurochrome injected intravenously an extremely valuable method of combating

infections—local and general—and infectious diseases had been produced.

J. H. Hill took up briefly the experimental work with mercurochrome. She discussed especially the reactions, believing this to be twofold. When the drug comes immediately in contact with the organism, it is a direct germicide. But when it is given intravenously and a lesion far from the site of injection is healed, undoubtedly the direct action is much less important than the indirect action, that is, the effect upon the host and his defenses. This is somewhat parallel to foreign protein injection, although different in many ways.

As proof of the curative action of intravenous mercurochrome, experiments were cited in the treatment of pneumococcus septicemia in rabbits, of anthrax in guinea-pigs, of typhoid cholecystitis in rabbits, of experimental syphilis in rabbits and of abscesses in guinea-pigs. In all of these it was shown that definite curative action could be obtained, either in every case, as in the treatment of wounds, cholecystitis and syphilis, or with a much higher percentage of recoveries than in the untreated controls, as in the pneumococcus septicemia and anthrax series.

The possible anaphylactoid reaction of mercurochrome intravenously had been checked according to the methods of Hanzlik and no such constriction of the bronchioles, distension of the lungs, hemorrhages or thrombi could be found after 5 mg./kg. doses of mercurochrome as Hanzlik found after the use of a wide variety of substances.

It should be possible by considering all of the facts, the different actions of mercurochrome locally and intravenously, to bring together and correlate our information and so to do away with the differences of opinion about this drug which have hitherto existed.

PROCEEDINGS OF ACADEMY MEETINGS, OCTOBER, 1926

Stated Meetings

Thursday Evening, October 7

ORDER

I. Executive Session.

II. Addresses.

a. The treatment of surgical tuberculosis, Sir Henry Gauvain, London (by invitation).

b. Relations of heart and lung in diseased conditions as observed by the fluoroscope, Edouard Rist, Paris (by invitation).

c. Periarteritis nodosa, Francis Harbitz, Oslo (by invitation).

Thursday Evening, October 21

ORDER

I. Executive Session.

a. Action on proposed amendment to Article III, Section 2 of the Constitution, introduced at the Stated Meeting of May 6th, 1926, to increase the resident fellowship. The amended article to read: "The number of Fellows residing within twenty-five miles of New York City Hall shall be limited to seventeen hundred and the number of Fellows outside the twenty-five mile limit shall be limited to four hundred."

b. Action on resolution proposed at Stated Meeting of May 6th, 1926: "Resolved: That the President be authorized to appoint a special committee to confer with representatives of the press on the possibility of the Academy's rendering assistance in giving out trustworthy statements on medical matters and that this resolution be transmitted to the Academy at its next meeting."

II. Lecture.

Observations during the period of under-nutrition in Germany, Friedrich von Müller, Munich (by invitation).

The lecture is published in this number of the Bulletin.

Section Meetings

Section of Pediatrics—Combined Meeting of the Section with The Philadelphia Pediatric Society and the New England Pediatric Society

Boston, Mass., October 16

PROGRAM

10:00–11:30 A.M., Children's Hospital Amphitheatre, 300 Longwood Avenue.

1. Some notes on infant feeding, Kenneth D. Blackfan, Joseph Johnston.
2. Some observations on the acid metabolism of intoxicated infants, Bengt Hamilton.
3. Variations in the diastase of the blood of infants, George Guest.
4. Reflex activity after transection of the spinal cord, Bronson Crothers.

11:45–12:30 P.M., Deaconess Hospital, Pilgrim Road.

1. Diabetes in childhood, Elliott P. Joslin.

12:45 P.M., Luncheon at the Harvard Medical School.

2:00–3:00 P. M., Peter Bent Brigham Hospital.

1. Neurological surgery in childhood, Harvey Cushing.

Section of Ophthalmology

Monday Evening, October 18

ORDER

I. Reading of the Minutes.

II. Clinical Cases.

a. Unilateral exophthalmos with choked disc. Probable tumor of the optic nerve, L. W. Crigler.

b. 1. Orbital neoplasm. Radium treatment. Result after three years' observation.
2. Case of high myopia. Extraction of lens with excellent visual results, H. H. Tyson.

c. Lymphangioma of orbit. Removed by Kronlein operation. Lantern slides, Martin Cohen.

d. An apparently cured case of glioma of the retina, Mark J. Schoenberg.

e. Case of retinal detachment with unusual feature, Clyde E. McDannald.

III. Paper of the Evening.

Classification of retinal diseases. Retinitis externa and choroiditis, Manuel U. Troncoso (by invitation).

Discussion, Mark J. Schoenberg.

Section of Obstetrics and Gynecology

Tuesday, October 26

I. Reading of the Minutes.

II. Case Reports.

Case reports of pregnancy following irradiation for menstrual disturbance, A. J. Rongy.

III. Paper of the Evening.

The effect of roentgen rays upon the ovary, an experimental and morphologic study, with clinical deductions, M. R. Robinson.

Discussion by Harold Bailey, Harvey Matthews (by invitation).

LIBRARY NOTES

Acquisition of Older Books

It may be of some interest to Fellows of the Academy to hear about the older books which have been added to the library between January 1st and September 15th. Three of the most valuable books in the list given below were purchased for the sum of $344.43 from a special fund which was subscribed to by some of the Fellows. Some of the other books came to the Academy through exchange or by gift and the remaining twenty-five cost $352.86. One of these latter books (Bartholomaeus Anglicus, *De proprietatibus rerum*, 1483) is a very important incunabulum giving us an idea of the biological and medical knowledge of the Middle Ages and cost $145.55, but even when we include this

one, the cost of these twenty-five old important books amounts to only three per cent. of the cost of the new books which have been received and of the periodicals which have arrived so far this year.

RECENT ACCESSIONS TO THE LIBRARY

New Books

Adams, H. Sanitary science.
Lond. Chapman. 1926. 188 p.

Arvedson, J. Medical gymnastics and massage in general practice. 2. ed.
Lond. Churchill. 1926. 284 p.

Bainbridge, J. S. Diet for the million.
Lond. Williams. 1926. 241 p.

Bernhard, O. Griechische und römische Münzbilder in ihren Beziehungen zur Geschichte der Medizin.
Zürich. Füssli. 1926. 93 p.

Bertwistle, A. P. & Shenton, E. W. H. A descriptive atlas of visceral radiograms.
Lond. Kimpton. 1926. 250 p.

Boas, I. & Kelling, G. Diätetik der Magen- und Darmkrankheiten.
2. Aufl. Leip. Thieme. 1926. 242 p.

Borchers, E. Allgemeine und spezielle Chirurgie des Kopfes.
Berlin. Springer. 1926. 382 p.

Cameron, H. C. Diseases of children.
Lond. Oxford pr. 1926. 199 p.

Carr-Saunders, A. M. Eugenics.
Lond. Williams. 1926. 256 p.

Cochrane, W. A. Orthopaedic surgery.
Edinburgh. Livingstone. 1926. 528 p.

Cock, R. Genesis v. evolution.
Lond. Stock. 1926. 77 p.

Cokkinis, A. J. Mesenteric vascular occlusion.
Lond. Baillière. 1926. 159 p.

Coster, G. Psycho-analysis for normal people.
Lond. Oxford pr. 1926. 232 p.

Crew, T. Health compendium and health publicity.
Leicester. Reader co. 1926. 260 p.

Deaver, J. B. Surgical anatomy of the human body.
2. ed. v. 1. Phila. Blakiston. 1926.

Dock, L. L. & Quimby, J. C. Text-book of materia medica for nurses.
8. ed. N. Y. Putnam. 1926. 317 p.

Dorland, W. A. N. The x-ray in embryology and obstetrics.
Lond. Kimpton. 1926. 420 p.

Eagleton, W. P. Cavernous sinus thrombophlebitis.
N. Y. Macmillan. 1926. 196 p.

Emanuel, J. Auricular fibrillation.
Birmingham. Cornish. 1926. 31 p.

Fano, G. Brain and heart.
Lond. Oxford pr. 1926. 142 p.

Gregory, H. H. C. Infant welfare.
Lond. Lewis. 1926. 144 p.

Grünwald, L. Lehrbuch der Kehlkopfkrankheiten. 3. Aufl.
München. Lehmann. 1925. 312 p.

Guibé, M. & Quénu, J. Chirurgie de l'abdomen. 6. éd.
Paris. Masson. 1926. 388 p.

Hajek, M. Pathology and treatment of the inflammatory diseases of the nasal accessory sinuses. 5. ed.
St. Louis. Mosby. 1926. 2 vols.

Handbuch der experimentellen Therapie, Serum- und Chemotherapie. Hrsg. von A. Wolff-Eisner. 2. Aufl.
München. Lehmann. 1926. 790 p.

Harris, D. T. Practical histology. 2. ed.
Lond. Lewis. 1926. 35 p.

Hazlitt, V. Ability: a psychological study.
Lond. Methuen. 1926. 147 p.

Heine, P. Kompendium der Milchuntersuchung für Tierärzte.
Hannover. Schaper. 1925. 102 p.

Hernaman-Johnson, F. Radiotherapy in relation to general medicine.
Lond. Oxford pr. 1926. 211 p.

Homburger, A. Vorlesungen über Psychopathologie des Kindesalters.
Berlin. Springer. 1926. 852 p.

Kawamura, R. Studies on tsutsugamushi disease.
Cincin. Coll. med. Univ. of Cincin. 1926. 229 p.

Kelley, I. V. Text-book of nursing technique.
Phila. Saunders. 1926. 366 p.

Lecène, P. & Leriche, R. Thérapeutique chirurgicale.
Paris. Masson. 1926. 3 vols.

Leonard, W. A. Organization and administration of the public health department.
Lond. Pitman. 1926. 148 p.

Linnekogel, H. Die Behandlung der Tuberkulose . . . mit Calcium-Silicum . . . durch Inhalation.
München. Lehmann. 1925. 106 p.

Lloyd, D. J. Chemistry of the proteins.
Lond. Oxford pr. 1926. 279 p.

Lodge, Sir O. Evolution and creation.
N. Y. Doran. 1926. 160 p.

McBride, P. The riddle of personality.
Lond. Heinemann. 1926. 190 p.

Mercier, C. Criminal responsibility.
N. Y. Phys. & surg. book co. 1926. 256 p.

Monrad-Krohn, G. H. The clinical examination of the nervous system. 3. ed.
Lond. Lewis. 1926. 201 p.

Montague, J. F. The modern treatment of hemorrhoids.
Phila. Lippincott. 1926. 296 p.

Morton, D. Invalid diet.
Lond. Heinemann. 1926. 98 p.

Mullowney, J. J. The hygiene of the home.
Bost. Christopher. 1926. 195 p.

National industrial conference board, inc. Medical care of industrial workers.
N. Y. Nat. ind. conf. bd. 1926. 112 p.

Neave, F. G. The law relating to injuries to workmen. 3. ed.
Lond. Wilson. 1926. 160 p.

Neumann, R. Atlas der radikal-chirurgischen Behandlung der Paradentosen.
Berlin. Meusser. 1926. 84 p.

Nolen, W., Hymans v. d. Bergh, A. A. [et al.]. Lebensversicherungs-Medizin.
Berlin. Springer. 1926. 368 p.

v. Noorden, V. & Isaac, S. Verordnungsbuch und diätetischer Leitfaden für Zuckerkranke. 3.–4. Aufl.
Berlin. Springer. 1926. 135 p.

Pannett, C. A. The surgery of gastro-duodenal ulceration.
Lond. Oxford pr. 1926. 154 p.

Paul, G. P. A text-book of materia medica for nurses. 5. ed.
Phila. Saunders. 1926. 352 p.

Porteus, S. D. & Babcock, M. E. Temperament and race.
Bost. Badger. 1926. 364 p.

Pryke, Sir Wm. [et al.]. How to live long.
Lond. Hutchinson. [1926.] 127 p.

Remington's practice of pharmacy. 7. ed.
Phila. Lippincott. 1926. 2090 p.

Richardson, F. H. Parenthood and the newer psychology.
N. Y. Putnam. 1926. 200 p.

Riddell, J. R. Handbook of medical electricity and radiology.
Edinb. Livingstone. 1926. 239 p.

Rievel, H. Handbuch der Milchkunde.
Hannover. Schaper. 1926. 432 p.

Rost, G. A. Hautkrankheiten.
Berlin. Springer. 1926. 405 p.

Rows, R. G. & Bond, W. E. Epilepsy.
Lond. Lewis. 1926. 138 p.

Scheidt, W. Allgemeine Rassenkunde. v. 1.
München. Lehmann. 1925. 585 p.

Schilder, P. & Kauders, O. Lehrbuch der Hypnose.
Wien. Springer. 1926. 110 p.

Siegrist, A. Refraction und Akkommodation des menschlichen Auges.
Berlin. Springer. 1925. 148 p.

Skramlik, E. von. Handbuch der Physiologie der niederen Sinne. v. 1.
Leip. Thieme. 1926.

Smith, W. J. A medical and surgical help for shipmasters and officers in the merchant navy. 6. ed.
Lond. Griffin. 1926. 337 p.

Stopes, M. C. The human body.
Lond. Gill pub. co. 1926. 224 p.

Sünner, P. The brain and the mind.
Lond. Allen. 1926. 112 p.
Thomson, J. A. Heredity. 5. ed.
Lond. Murray. 1926. 542 p.
Townsend, H. Social work: a family builder.
Phila. Saunders. 1926. 247 p.
Transactions of the American laryngological association. 1925.
Transactions of the College of physicians of Philadelphia. 1925.
Watson, J. K. A handbook for nurses. 7. ed.
Lond. Faber. 1926. 802 p.
Wilkinson, W. C. The principles of immunity in tuberculosis.
Lond. Nisbet. 1926. 141 p.

Older Books

(acquired between january 1 and september 15, 1926)

An account of the New York Hospital.
N. Y. Collins & Co. 1811. 65 p.
Albertus Magnus. Alberti Magni drei bücher von wunder. . . .
[Frankfurt a. M. Egenolph] 1537. 19 l.
Astruc, Jean. Tractatus pathologicus.
Genevae. Cramer . . . 1743. 232 p.
Aubrey, John. Miscellanies upon various subjects. . . .
5. ed. Lond. Reeves & Turner. 1890. 221 p.
Bartholomaeus, Anglicus. De proprietatibus rerum.
[Nurenberge. A. Koburger. 1483] unp.
Blaine, Delabere. Canine pathology.
4. ed. Lond. Longman, Orme Co. 1841. 324 p.
Boerhaave, Hermannus. Institutiones et experimenta chemiae.
Venetiis. S. Coleti. [1726].
v. 1—292 p.
v. 2—350 p.
Boyle, Robert. Of the reconcileableness of specifick medicines. . . .
Lond. S. Smith. 1685. 225 p.
Bridges, J. H. Life and work of Roger Bacon.
Lond. Williams & Norgate. 1914. 173 p.
Brown, John. Horae subsecivae. Locke & Sydenham. . . .
Edinburgh. D. Douglas. 1890. 468 p.

Browne, (Sir) Thomas. Certain miscellany tracts.
Lond. C. Mearne. 1684. 215 p.

Browne, (Sir) Thomas. Posthumous works. . . .
Lond. Curll. 1712. var. p.

Caius, Johannes. The works of Jean Caius, M.D. . . .
Cambridge, [Eng.] Univ. Pr. 1912. unp.

Cassal, J. Traicté de la pesté. . . .
Lyon. B. Rigaud. 1589. p. 55.

Celsus, Aurelius Cornelis. De medicina.
[Florentiae. a Nicolao imp. 1478] var. p.

Cheyne, George. An essay on regimen—five discourses.
3. ed. Lond. D. Browne. 1753. 344 p.

Cockburn, William. The nature and cure of fluxes.
3. ed. Lond. J. Clarke. 1724. 344 p.

Digby, (Sir) Kenelm. Powder of sympathy. . . .
3. ed. Lond. R. Lowndes. 1660. 152 p.

Eliot, Walter Graeme. Portraits of the noted physicians of New York.
[N. Y. 1900] 200 p.

Evening thoughts. By a physician.
Lond. J. Van Voorst. 1850. 144 p.

Fabricius ab Aquapendente, Hieronymus. L'opere chirurgiche. . . .
Bologna. G. Longhi. 1678. 359 p.

Ferguson, John. Bibliotheca chemica. . . .
Glasgow. J. Maclehose & sons. 1906.
v. 1—487 p.
v. 2—598 p.

Fleming, George. Pasteur and his work. . . .
Lond. W. Clowes & sons. 1866. 75 p.

Franklin, Alfred. La vie privée d'autrefois. Les médecins.
Paris. E. Plon. 1892. 305 p.

Galen, Cladius. De naturalibus facultatibus libri tres. . . .
Lugduni. apud G. Rovillium. 1550. 205 p.

Gesner, Conrad. Bibliotheca instituta et collecta. . . .
Tigri. apud Christophorum Froschoverum. 1574. 691 p.

Gilbert, William. De magnete. . . .
Lond. P. Short. 1600. 240 p.

Gunther, Robert Theodore. Early science in Oxford.
Oxford. 1923. 407 p.

Haller, Albertus. Physiology; being a course of lectures. . . .
2. ed. Lond. G. Robinson. 1772.
v. 1—339 p.
v. 2—498 p.

Highmore, Nathanael. The history of generation. . . .
Lond. J. Martin. 1631. 141 p.

Kennett, White. Bibliothecae Americanae primordia.
Lond. J. Churchill. 1713. 275 p.

Kipling, Rudyard. Doctors. An address delivered to the students. . . . Middlesex hospital. . . .
Lond. Macmillan & co. 1908. 31 p.

Laurentius, Andreas. Toutes les oeuvres. . . .
Rouen. J. Berthelet. 1661. 572 p.

de Leon, Andres. Practico de morbo gallico. . . .
Valladolid. Sanche. 1605. 126 p.

Life (The) and letters of Charles Darwin. . . .
Lond. J. Murray. 1887.
v. 1—395 p.
v. 2—393 p.
v. 3—418 p.

Mesue *Damascenus,* Joannes. De morbis internis curandis liber.
Lugundi. A. Vincentium. 1551. 493 p.

Moore, Charles Hewitt. On going to sleep.
Lond. R. Hardwicke. 1868. 60 p.

Nightingale, Florence. Notes on nursing: what it is and what it is not.
Lond. Harrison. [1860] 79 p.

Phinney, H. F. The water cure in America. . . .
2. ed. N. Y. J. Wiley. 1849. 288 p.

Poems and songs of patriotism and war. Ed. by Charles L. Dana.
Woodstock, Vt. Elm Tree Pr. [1918] 35 p.

Portius, Franciscus. Medica decas, ejusdem authoris. . . .
Lutetiae Parisiorum. M. Mondiere. 1613. 462 p.

Quincy, John. The dispensatory of the Royal College of Physicians in London. . . .
2. ed. Lond. R. Knaplock. 1727. 362 p.

Robinson, Bryan. Observations on the virtues and operations of medicines.
Dublin. Ewing, Smith & Faulkner. 1752. 216 p.

Shapter, Thomas. The history of cholera in Exeter in 1832.
Lond. J. Churchill. 1849. 297 p.

Smith, Eustace. On wasting disease of infants and children.
4. ed. Lond. J. & A. Churchill. 1884. 372 p.

Sylvius [de la Boë] Franciscus. Institutie medica. . . .
[s. l. 1770?] 310 p.

Sylvius [de la Boë] Franciscus. Trajecti ad Rhenum. G. v. d. Water. 1695.
v. 1—582 p.
v. 2—934 p.

Tennent, John. Physical enquiries.
Lond. T. Gardner. 1742. 69 p.

Tissot, Simon André D. L'onanisme. . . .
Nouv. ed. Paris. Bossange, Masson & Besson. 1805. 162 p.

Tweedie (Mrs.) Ethel. George Harley, F.R.S. The life of a London physician. . . .
Lond. Scient. Press. 1899. 360 p.

Warren, Edward. Boyleston prize dissertations. . . . 1838 and 1839.
Phila. A. Waldie. 1840. 122 p.

Willis, Thomas. Pathologicae cerebri et nervosi. . . .
Amstelodami. Elzevirium. 1668. 338 p.

CEREMONIES IN CONNECTION WITH THE OPENING OF THE NEW BUILDING OF THE ACADEMY

A dinner in honor of the new Honorary Fellows will be given at the Waldorf-Astoria Hotel on Wednesday, November 17, at 7 p. m. Addresses will be delivered by Dr. Samuel A. Brown, President, The New York Academy of Medicine, and several of the Honorary Fellows.

The opening exercises at the new building will be held on Thursday, November 18, at 3.30 p. m., Fifth Avenue and One Hundred and Third Street.

Invocation by Rev. Henry Sloane Coffin, D.D.

Addresses: The Academy—its Services in Medical Progress, Dr. Samuel A. Brown, President, The New York Academy of Medicine. The Building of the Academy, Dr. Arthur B. Duel, Chairman, Building Committee.

The Fellows and their friends are invited to attend a reception and inspection of the building to be held on Thursday, November 18, from 4.00 to 6.00 p. m., Fifth Avenue and One Hundred and Third Street.

The first meeting to be held in the new building will be a Stated Meeting of the Academy in Hosack Hall, Fifth Avenue and One Hundred and Third Street, Thursday, November 18, at 8.30 p. m. At this meeting there will be an election of Honorary Fellows and the Carpenter Lecture will be delivered by Michael I. Pupin, of Columbia University, on "Ionization and Chemical Reactions."

ACTION ON INCREASED MEMBERSHIP AND THE RELATIONS OF THE ACADEMY TO THE PRESS

On May 6 a resolution was introduced at a Stated Meeting of the Academy, to amend the Constitution so as to increase the number of Fellows residing within 25 miles of the City Hall, New York City.

At a meeting on October 21, the Academy voted to increase the number of resident Fellows from 1,625 to 1,700.

During the Spring, a number of discussions were held at meetings of the Council of the Academy as to whether or not it would be proper for the Academy to issue statements to the press on medical matters, which would be of educational value to the public. No definite plan was considered except that if such a plan were agreed upon by the Academy, statements would have to be made in the name of the Academy and not in the name of any of the Fellows.

The Council recommended to the Academy that the matter be considered, and the following resolution was introduced at a Stated Meeting on May 6:

> RESOLVED: that the President be authorized to appoint a special committee to confer with representatives of the press on the possibility of the Academy's rendering assistance in

> giving out trustworthy statements on medical matters and that this resolution be transmitted to the Academy at its next meeting,

and at a meeting of the Academy on October 21 it was moved and seconded that the matter be laid on the table.

It was then pointed out that the adoption of a resolution would not in any way complicate the situation, nor would it authorize the President or the Council to take any specific action,—the resolution only authorized the President to appoint a special committee to confer with the representatives of the press. It was evident that the Fellows were opposed to any discussion of the matter and the motion to table was adopted.

CANDIDATES RECOMMENDED FOR ELECTION TO FELLOWSHIP OCTOBER 6, 1926

Rudolph Boenke, M.D., 320 Grand Avenue, L. I. City.
Samuel Gitlow, M.D., 865 Walton Avenue.
Roy Biggs Henline, M.D., 901 Lexington Avenue.
Edward Crane Lyon, Jr., M.D., 100 East 66th Street.
John Sturdivant Read, M.D., 174 Clinton Street, Brooklyn.

CANDIDATES RECOMMENDED FOR ELECTION TO ASSOCIATE FELLOWSHIP

Charles Francis Bodecker, D.D.S., 576 Fifth Avenue.
Homer Folks, A.B., 105 East 22nd Street.
Edgar Grim Miller, Jr., Ph.D., 180 Claremont Avenue.
Oliver Smith Strong, Ph.D., Livingston Hall, Columbia Univ.
Frank Thorn Van Woert, M.D.S., 302 East 35th Street.

DEATHS OF FELLOWS OF THE ACADEMY

Joshua Lindley Barton, M.D., 117 East 61st Street, New York, N. Y., graduated in medicine from College of Physicians and Surgeons, New York City, 1881; elected a Fellow of the Academy December 5, 1889; died September 10, 1926.

William Francis Campbell, A.B., M.D., 394 Clinton Avenue, Brooklyn, New York, graduated in medicine from Long Island College Hospital, 1892; elected a Fellow of the Academy June 5,

1902; died September 7, 1926. Dr. Campbell was a Fellow of the American Medical Association, a Fellow of the American College of Surgeons, a member of the American Surgical Society and a member of the American Anatomical Society.

Franklin Abbott Dorman, A.B., A.M., M.D., 121 East 60th Street, New York, N. Y., graduated in medicine from College of Physicians and Surgeons, New York City, 1898; elected a Fellow of the Academy May 5, 1904; died August 6, 1926. Dr. Dorman was a Fellow of the American Medical Association, a Fellow of the American College of Surgeons and a member of the Obstetrical Society.

Arthur Middleton Jacobus, M.D., 131 West 70th Street, New York, N. Y., graduated in medicine from Bellevue Hospital Medical College, New York City, 1876; elected a Fellow of the Academy December 4, 1879; died September 18, 1926. Dr. Jacobus was a Fellow of the American Medical Association, a member of the Obstetrical Society and a member of the Society of Alumni of Presbyterian Hospital. He was also Attending Physician of Tuberculosis at Vanderbilt Clinic.

Frederick Samuel Mandlebaum, M.D., 1300 Madison Avenue, New York, N. Y., graduated in medicine from Bellevue Hospital Medical College, New York City, 1889; elected a Fellow of the Academy May 5, 1904; died August 7, 1926. Dr. Mandlebaum was a fellow of the American Medical Association, a member of the American Pathological and Bacteriological Society, a member of the American Cancer Research Institute, a member of the Pathological Society and a member of the Society of Associated Alumni of Mt. Sinai Hospital.

Charles Edward Nammack, Ph.D., M.D., 379 Park Avenue, New York, N. Y., graduated in medicine from Bellevue Hospital Medical College, New York City, 1881; elected a Fellow of the Academy May 1, 1884; died October 4, 1926. Dr. Nammack was a Fellow of the American College of Physicians, a member of the Society of Alumni of Bellevue Hospital, Visiting Physician at Bellevue Hospital, Consultant Physician at St. Vincent's Hospital, and Professor of Clinical Medicine, Cornell University Medical College.

Sanders McAllister Payne, M.D., Dumont, N. J., graduated in medicine from Vanderbilt University, Nashville, Tennessee, 1875; elected a Fellow of the Academy February 1, 1894; died September 11, 1926. Dr. Payne was a Fellow of the American College of Surgeons, and a member of the American Academy of Ophthalmology and Oto-Laryngology.

John Duncan Quackenbos, A.B., A.M., M.D., 823 West End Avenue, New York, N. Y., graduated in medicine from College of Physicians and Surgeons, New York City, 1871; elected a Fellow of the Academy October 16, 1884; died August 1, 1926. Dr. Quackenbos was a Fellow of the American Medical Association and a member of the American Society for the Advancement of Science.

August Seibert, M.D., 222 East 72nd Street, New York, N. Y., graduated in medicine from University of Leipzig, Germany, 1877; elected a Fellow of the Academy May 21, 1885; died July 29, 1926.

Edwin Holmes, A.B., M.D., Exeter, New Hampshire, graduated in medicine from the College of Physicians and Surgeons, New York City, in 1895; elected a Fellow of the Academy March 7, 1901; died October 15, 1926.

Robert Grigg Reese, M.D., 50 West 52nd Street, New York City, graduated in medicine from New York University in 1891; elected a Fellow of the Academy November 2, 1911; died October 18, 1926. Dr. Reese was a Fellow of the American Medical Association, a Fellow of the American College of Surgeons, a member of the American Ophthalmological Society, a member of the American Academy of Ophthalmology and Oto-laryngology, a member of the Ophthalmological Society and a member of the Alumni Association of New York Eye and Ear Infirmary. He was Consulting Ophthalmologist at New York Infirmary for Women and Children, at Memorial, Nursery and Child's, and at Reconstruction and Bellevue hospitals. He was Professor of Ophthalmology at Cornell University Medical College.

Robert Justice Wilson, B.S., M.D., 111 East 10th Street, New York City, graduated in medicine from University and Bellevue

Hospital Medical College, New York City, in 1890; elected a Fellow of the Academy March 1, 1906; died October 16, 1926. Dr. Wilson was a Fellow of the American Medical Association, a member of the National Tuberculosis Association, a member of the American Bacteriological Society, a member of the Society of Alumni of Bellevue Hospital and a member of the Pathological Society.

DONATIONS TO THE LIBRARY FUNDS

Donations and bequests are solicited by The New York Academy of Medicine for the maintenance and expansion of the Library.

A donation or bequest of $5,000 or more will provide for a special library fund, the income of which may be used for the general purposes of the Library or restricted to the purchase of books and periodicals, as the donor or testator may indicate. When so restricted, a special bookplate will be used. A donation or bequest of less than $5,000 but more than $1,000 will provide for a named library fund, the income of which will be used for general library purposes or restricted for the purchase of books and periodicals, as desired by the donor or testator.

Donations of less than $1,000 will be added to the general library funds.

FORM OF BEQUESTS

The following is a brief legal form as a suggestion under which bequests may be made in behalf of the Academy:

I give, devise and bequeath unto "The New York Academy of Medicine" of the City of New York, State of New York, a corporation duly incorporated by the Legislature of the State of New York by an act, entitled "An act to incorporate The New York Academy of Medicine," passed June 23, 1851, and amended June 4, 1853, June 2, 1877, and April 24, 1925.....

DATES OF MEETINGS

Stated Meetings of the Academy, 1st and 3rd Thursdays.
Trustees, 2nd Wednesday.

Council, 4th Wednesday.
Committee on Library, 2nd Tuesday.
Committee on Admission, 1st Wednesday.
Public Health Committee, Mondays.
Committee on Medical Education, 2nd Thursday.
Building Committee, 1st and 3rd Tuesdays.
Publication Committee, date varies.
Program Committee, date varies.

DATES OF SECTION MEETINGS

Dermatology and Syphilis, 1st Tuesday.
Surgery, 1st Friday.
Neurology and Psychiatry, 2nd Tuesday.
Pediatrics, 2nd Thursday.
Otology, 2nd Friday.
Ophthalmology, 3rd Monday.
Medicine, 3rd Tuesday.
Genito-Urinary Surgery, 3rd Wednesday.
Orthopedic Surgery, 3rd Friday.
Obstetrics and Gynecology, 4th Tuesday.
Laryngology and Rhinology, 4th Wednesday.
Historical and Cultural Medicine, date varies.

COMMITTEES OF THE ACADEMY

Committee on Admission

JOHN J. MOORHEAD, *Chairman* — GEORGE B. WALLACE
FREDERICK T. VAN BEUREN, JR. — ARTHUR M. WRIGHT
SAMUEL A. BRADBURY

Committee on Library

FRANK J. BLODGETT, *Chairman* — J. RAMSAY HUNT
CHARLES A. ELSBERG — RUSSELL L. CECIL
ERNEST G. STILLMAN

Committee on Public Health Relations

CHARLES L. DANA, *Chairman*

Executive Committee

ROBERT J. CARLISLE — JAMES A. MILLER — W. GILMAN THOMPSON
JAMES B. CLEMENS — BERNARD SACHS — PHILIP VAN INGEN
LEWIS F. FRISSELL — FREDERIC E. SONDERN — GEORGE B. WALLACE
JOHN A. HARTWELL — GEO. DAVID STEWART — E. H. LEWINSKI-CORWIN, *Executive Secretary*

Committee on Medical Education

JAMES F. MCKERNON, *Chairman*

Executive Committee

ARTHUR F. CHACE — HOWARD FOX — SAMUEL J. KOPETZKY
CHARLES A. ELSBERG — ISIDORE FRIESNER — JOHN J. MOORHEAD
HAVEN EMERSON — JOHN E. JENNINGS — JOHN O. POLAK
NELLIS B. FOSTER — ARNOLD KNAPP — FREDERICK P. REYNOLDS, *Medical Secretary*

GRACE CARSTENSEN, *Executive Secretary*

Building Committee

ARTHUR B. DUEL, *Chairman* — SIGISMUND S. GOLDWATER
SAMUEL A. BROWN — SETH M. MILLIKEN
CLARENCE C. BURLINGAME — WALTER L. NILES
CHARLES A. ELSBERG — FREDERICK T. VAN BEUREN, JR.
ARTHUR M. WRIGHT

Nominating Committee

LEWIS F. FRISSELL, *Chairman*
EDWIN BEER — J. BENTLEY SQUIER

Committee on Publication

CHARLES L. DANA, *Chairman* — E. LIVINGSTON HUNT
FENWICK BEEKMAN — ALBERT R. LAMB
SAMUEL A. BROWN — FREDERIC E. SONDERN
CHARLES A. ELSBERG — GEORGE B. WALLACE

Program Committee

SAMUEL A. BROWN — NELLIS B. FOSTER
HERBERT S. CARTER — HOWARD FOX
CHARLES A. ELSBERG — FREDERICK T. VAN BEUREN, JR.

Officers of Sections

Dermatology and Syphilis
MIHRAN B. PAROUNAGIAN, *Chairman*
GEORGE C. ANDREWS, *Secretary*

Medicine
DAVENPORT WEST
HENRY JAMES

Surgery
FREDERICK W. BANCROFT
MORRIS K. SMITH

Genito-Urinary Surgery
FREDERICK T. LAU
STANLEY R. WOODRUFF

Neurology and Psychiatry
THOMAS K. DAVIS
WALTER M. KRAUS

Orthopedic Surgery
HARRY FINKELSTEIN
ARTHUR KRIDA

Pediatrics
LOUIS C. SCHROEDER
WILLIAM ROPES MAY

Obstetrics and Gynecology
HARBECK HALSTED
HERVEY C. WILLIAMSON

Otology
J. L. MAYBAUM
R. T. ATKINS

Laryngology and Rhinology
JOHN E. WELCH
ARTHUR NILSEN

Ophthalmology
CLYDE E. MCDANNALD
THOMAS H. CURTIN

Historical and Cultural Medicine
L. PIERCE CLARK
CHARLES E. ATWOOD

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